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Mycoplasmal Pneumonia Associated With Abscess Of the Lung

JAMES E. LEWIS, M.D., AND CHARLES SHEPTIN, M.D., *San Jose*

THE RADIOLOGIC CHANGES in mycoplasmal pneumonia are well documented.[1-5] The infiltrate is usually unilateral and involves one or more segments of a single lobe, usually one of the lower lobes. A soft, patchy, localized bronchopneumonia is usually seen which frequently fans out from the hilus of the lung. Occasionally, a mottled, reticular or disseminated pneumonia is seen; lobar pneumonia is uncommon. Pleural effusions are generally infrequent and small,[5,7-9] although massive effusions have been reported.[5,6] A review of the literature failed to reveal any case of documented lung abscess in an uncomplicated case of mycoplasmal pneumonia.

Report of a Case

The patient, a 24-year-old Caucasian auto body shop worker, presented with a two-week history of sore throat followed by substernal and anterolateral chest pain. The patient produced copious amounts of green, mucoid, non-fetid sputum that was occasionally blood-tinged. Symptoms were intermittent but progressive with headache, chills, diaphoresis, malaise and myalgias. He had lived in Bakersfield, California, in the past years but denied contact with tuberculosis or pathogenic inhalants except for the smoking of one pack of cigarettes a day. He consumed 12 to 24 ounces of beer a day and was allergic to penicillin.

On examination at Santa Clara Valley Medical Center on October 3, 1970, his temperature was 99.4°F., blood pressure 112/78 mm of mercury, pulse rate 96, and respiration rate 24. His pharynx was mildly erythematous but without exudate. He had moderate respiratory distress and bilateral splinting on inspiration but no dyspnea or cyanosis. Dullness and decreased breath sounds were noted over the base of the left lung posteriorly, with occasional fine moist rales.

The roentgenologic examination of the chest on admission revealed a homogeneous infiltrate in the superior division of the lingular segment (Figure 1). In the lateral aspect of the lingula there was an air-fluid level consistent with pulmonary cavitation. Four days later the effusion had progressed (Figure 2). Following pleural biopsy with the Abrams needle, 450 ml of serosanguinous fluid was removed. Before the biopsy the fluid was serous. Analysis of the fluid showed a total protein of 5.4 gm per 100 ml, a red blood cell count of 6,000, neutrophils 2,400 and mononuclear cells 2,400 per cu mm. Aerobic and anaerobic cultures of the pleural fluid were negative for bacteria, acid-fast organisms and fungi.

Leukocytes numbered 10,800 per cu mm, with 84 percent neutrophils, 12 percent lymphocytes, 3 percent monocytes, and 1 percent eosinophils. The hematocrit was within normal limits. Screening chemical panel 16 was normal except for serum albumin of 2.9 gm per 100 ml and glutamic oxaloacetic acid transaminase 110 mμ per ml (normal 10 to 50). Immunodiffusion of the patient's serum revealed an immunoglobulin A (IgA) level of 408 mg per 100 ml (normal 90 to 274 mg), IgG of 1500 mg (normal 737-1481) and IgM of 160 mg (normal 40-128).

A Gram stain of the sputum showed a few pus cells and rare Gram-positive cocci and Gram-negative rods. Aerobic cultures of deep productive sputum before the administration of antibiotics and twice during the treatment with cephalosporins grew saprophytic neisseria, alpha hemolytic streptococci, hemophilus species, gamma hemolytic streptococci, diptheroids, and rare yeast colonies. Specific anaerobic cultures of the sputum were not obtained. Three sputum cultures for fungi showed no growth and three paired cultures of the blood for aerobic and anaerobic organisms were negative.

A coccidioidin skin test (1:100) was positive (20 mm induration at 72 hours), but complement fixation and precipitin antibody titers for coccidiomycosis were negative.* The tuberculin skin test (intermediate strength of purified protein

From the Department of Medicine, Santa Clara Valley Medical Center, San Jose.

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Reprint requests to J. E. Lewis, M.D., Department of Medicine, Santa Clara Valley Medical Center, 751 S. Bascom Avenue, San Jose, Ca. 95128.

*Performed by the laboratories of Dr. D. Pappagianis, University of California, Davis, School of Medicine, Davis, California.

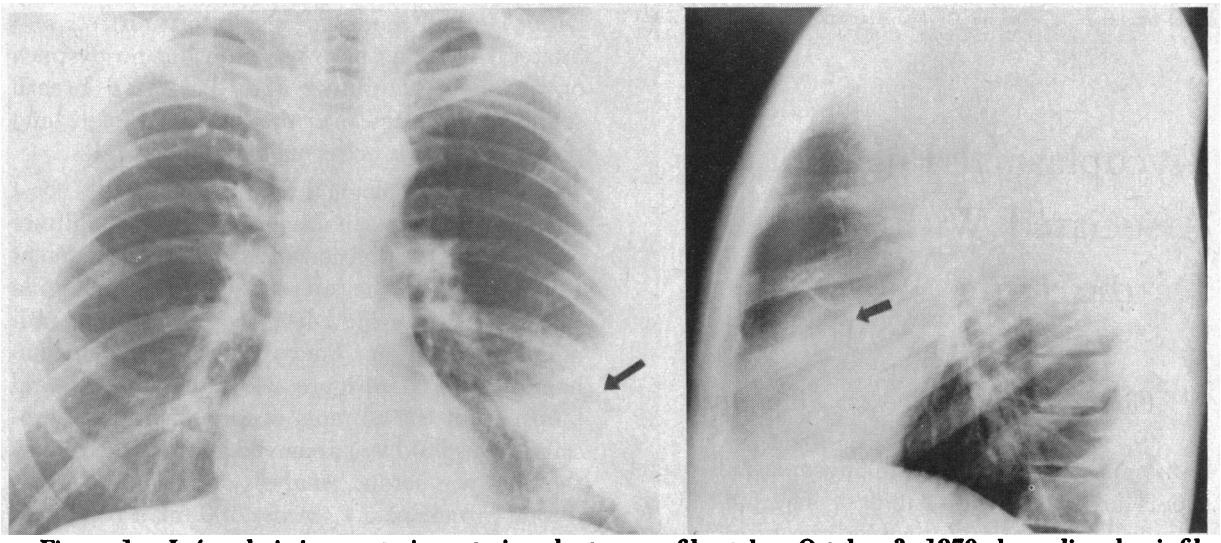

Figure 1.—*Left*, admission posterior-anterior chest x-ray film taken October 3, 1970 shows lingular infiltrate with air fluid level in the lateral portion consistent with cavitation. Peribronchial infiltrate seen in the right lower lobe. *Right*, lateral view demonstrates cavitation and associated fluid in the major fissure.

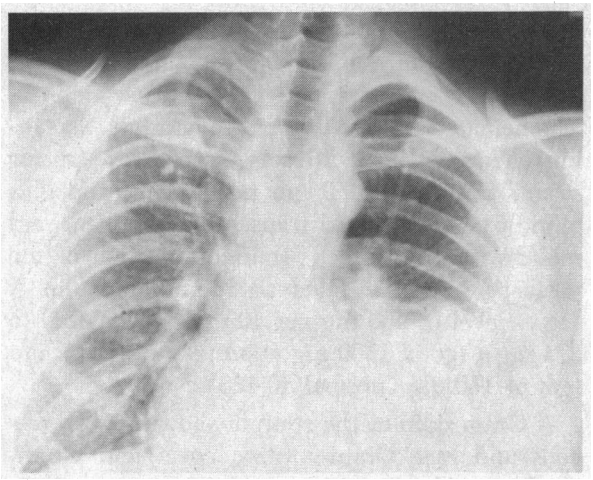

Figure 2.—Film taken October 7, 1970, shows massive left pleural effusion and left lower lobe infiltrate obscuring the cavity.

derivative) was normal and sputum smears and cultures for acid-fast bacilli were negative.

Complement fixation titers** were initially less than 1:8 for influenza A, influenza B, adenovirus and Q fever. Psittacosis-lymphogranuloma venereum titer was 1:8 ("indicating infection at some time") and the titer of complement fixation antibody for mycoplasma pneumoniae was 1:1024. Titers during convalescence obtained three weeks later and performed simultaneously were unchanged except for Mycoplasma pneumoniae which had decreased to a 1:8 titer. During this time interval a rise in cold agglutinin titers from less than 1:4 to 1:32 was noted.

The patient's fever, chest pain, productive cough and radiologic changes were unimproved after four days of treatment with cephalothin, 3 grams intravenously every six hours. On the fifth day, because of local venous tenderness, therapy was changed to cephaloridine, 1 gm intravenously every six hours. The pulse rate varied from 80 to 100, the respiratory rate from 18 to 26, and the oral temperature remained at 101°F. The lack of clinical response and the suspicion of mycoplasmal pneumonia led to the administration of tetracycline by mouth, 500 mg every six hours. Over the next 96 hours the patient became afebrile and improved clinically. By the sixteenth hospital day the chest x-ray showed resolution of the pleural fluid and parenchymal infiltrate. A small cavity was still present. Six weeks after admission a chest x-ray film revealed only minimal lingular scarring.

Comment

Diagnostic criteria for mycoplasmal pneumonia include: (a) isolation of Mycoplasma pneumoniae, (b) a four-fold rise in the convalescent titer of complement fixation antibody[1,5,9-11] or (c) a single complement fixation titer variously reported as greater than 1:32 to 1:256.[1,9,10] Although specific attempts at isolation of Mycoplasma

**Performed by the California State Department of Public Health, Berkeley, with the use of Microtiter Complement Fixation Test with "raw" and "lipid" antigen as described by Purcell RH, Chanock RM *In* Diagnostic Procedures for Viral and Rickettsial Infections, Ed Rennett, NJ Schmidt, eds, Chapter 23, American Public Health Association, Inc., New York, 1969, p. 814.

pneumonia were not carried out, the patient's initial titer for complement fixation antibody of 1:1024 three weeks after the onset of symptoms meets the most rigid criterion of the last standard, and indeed is one of the highest recorded in all of the above series. A four-fold rise in cold agglutinin titer,[3,4,10,12,13] as occurred in our patient, is considered strongly supportive of a diagnosis of Mycoplasma pneumoniae infection.

The association of Mycoplasma pneumoniae with lung abscess has not been previously documented. George et al[5,13] commented upon four patients who had "pneumatoceles" that cleared quickly. In the radiographs published by those observers the "pneumatoceles" (although admittedly not well seen) appeared to be outside the area of incomplete segmental consolidation and did not contain any fluid. In all probability they really were pneumatoceles and not true abscesses of the lung. In their series of 500 patients in 1951 with "primary atypical pneumonia," Robertson and Morle[8] mentioned two cases of abscess formation within the pulmonary infiltrate. Unfortunately, detailed bacteriologic study was not undertaken and no mention of cold agglutinin titers in the two patients was made. At that time, complement fixation titers for Mycoplasma pneumoniae were not available. Because of these reservations, the exact nature of the illness in the two patients with lung abscess is uncertain.

Pleural effusions, although infrequent, have been reported in association with mycoplasmal pneumonia.[5,7-9] Massive pleural effusions, as was present in the case reported, are distinctly rare.[5,6] Our patient's elevated pleural fluid protein and the mixed numbers of leukocytes and red blood cells are similar to those reported in other effusions associated with mycoplasmal pneumonia,[4,9] although the fluid for analysis was taken after the biopsy in the present case, making more detailed interpretation difficult. The pleural biopsy showed no evidence of granulomatous disease.

The positive coccidioidin skin test with negative complement fixation and precipitin antibody titers three weeks after the onset of symptoms is attributed to previous habitation in an endemic area with probable subclinical infection. The low positive Psittacosis-lymphogranuloma venerum titer was the same in acute and convalescent sera and may be attributed to previous subclinical infection. The elevations of the IgA, IgG, and IgM are nonspecific but are compatible with those previously described in Mycoplasma pneumoniae infections.[14]

The modest leukocytes without left shift, especially in view of the extensive pulmonary involvement and effusion, might favor viral rather than bacterial cause. A primary abscess of the lung cannot be excluded completely in our patient, because it would have a similar clinical course and defervescence as that of our patient, and specific anaerobic cultures of the sputum were not obtained. However, the absence of fetid breath and of musty odor to the sputum, of periods of unconsciousness, and of emesis or trauma are evidence against a primary abscess of the lung. The relatively rapid resolution of the cavity makes the diagnosis of a pre-existing cavity, cyst, or bleb with superimposed Mycoplasma pneumoniae infection unlikely. We cannot with certainty exclude some other primary pulmonary disorder that might predispose to the development of mycoplasmal infections. Our patient clearly had a mycoplasmal pneumonia with a definite cavity that contained fluid. The nonbacterial nature of the illness was further substantiated by failure to isolate pathogenic bacteria despite multiple cultures of the sputum, blood, and pleural fluid and gradual clinical and radiographic worsening in spite of intensive antibiotic therapy with the cephalosporins. The clearing of the pneumonia and abscess both clinically and radiographically, after the addition of tetracycline therapy, is also compatible with the well documented sensitivity of Mycoplasma pneumoniae to tetracycline.[13,15,16]

Summary

A previously undescribed complication of mycoplasmal pneumonia, the formation of a pulmonary abscess, is reported in one patient. The complement fixation titer for Mycoplasma pneumoniae was positive at 1:1024 and a four-fold rise in cold agglutinin titer was observed. All bacteriologic studies were normal. On radiologic examination a lingular infiltrate with an air-fluid level consistent with cavitation was demonstrated. Massive pleural effusion developed before the gradual resolution of both processes. Perhaps more careful study of patients with "primary abscess" of the lung may reveal additional cases of mycoplasmal pneumonia associated with lung abscess.

REFERENCES

1. Graystone JT, Foy HM, Kenny GE: Mycoplasma (PPLO) in human disease. Disease-a-Month. Dec 1967
2. Herbert DH: The roentgen features of Eaton agent pneumonia. Am J Roentg 98:300-4, 1966
3. Mufson MA, Manko MA, Kingston JR, et al: Eaton agent pneumonia—Clinical features. JAMA 178:369-374, 1961
4. Rytel MW: Primary atypical pneumonia: Current concepts. Am J Med Sci 247:84-104, 1964
5. George RB, Weill H, Rasch JR, et al: Roentgenographic appearance of viral and mycoplasmal pneumonias. Am Rev Resp Dis 96: 1144-1150, 1967
6. Decancq HC, Lee FA: Mycoplasma pneumoniae pneumonia. JAMA 194:1010-1011, 1965
7. Curnen EC, Mirick GS, Ziegler JE Jr, et al: Studies of primary atypical pneumonia—I. Clinical features and results of laboratory investigations. JCI 24:209-226, 1945
8. Robertson PW, Morle KDF: An explanation of the "primary atypical pneumonia" syndromes. Br Med J 2:994-998, 1951
9. Fine NL, Smith LR, Sheedy PF: Frequency of pleural effusions in Mycoplasma and viral pneumonias. N Engl J Med 283:790-793, 1970
10. Biberfield G, Stenbeck J, Johnsson T: Mycoplasma pneumoniae infections in hospitalized patients with acute respiratory illness. Acta Path et Microbiol Scand 74:287-300, 1968
11. Alexander ER, Foy HM, Kenny GE, et al: Pneumonia due to Mycoplasma pneumoniae—Its incidence in the membership of a cooperative medical group. N Engl J Med 275:131-136, 1966
12. Jordan WS, Albright RW, McCain FH, et al: Clinical variations in primary atypical pneumonia. Am J Med 10:3-20, 1951
13. George RB, Ziskind MM, Rasch JR, et al: Mycoplasma and adenovirus pneumonias—Comparison with other atypical pneumonias in a military population. Ann Int Med 65:931-942, 1966
14. Fernald GW, Clyde WA Jr, Denny FW: Nature of the immune response to Mycoplasma pneumoniae. J Immuno 98:1028-1038, 1967
15. Kingston JR, Chanock RM, Mufson MA, et al: Eaton agent pneumonia. JAMA 176:120-123, 1961
16. Clyde WA, Denny FW: The etiology and therapy of atypical pneumonia. Med Cl No Am 47:1201-1218, 1963

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Fascioliasis

Response to Bithionol

ROBERT G. KNODELL, M.D.,
EDWARD KIRSCH, M.D., AND
GEORGE C. RYGG, M.D., *San Francisco*

FASCIOLA HEPATICA is an increasingly important parasite of man in Latin American and Mediterranean countries. Although the organism is enzootic in extensive areas of the southern, southwestern, and western United States,[1] only six cases of human fascioliasis have been reported in this country.[1-5] The course of this disease is characterized by clinical manifestations for up to three months before the diagnostic appearance of ova in the stools. Our report illustrates the clinical features which should arouse suspicion of fascioliasis and describes the clinical course and changes in serial liver scans during therapy with bithionol.*

From the Department of Medicine, School of Medicine, University of California, San Francisco.

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Reprint requests to: Editorial Office, 998-M, Department of Medicine, University of California, San Francisco, Ca. 94122.

Report of a Case

A 55-year-old Mexican-American man was admitted to another hospital in April 1971. He had had constant right upper quadrant pain, occasional diarrhea, malaise, and fever of one week's duration. He and his family had returned from a one-month vacation in Mexico in February. Physical examination revealed tender hepatomegaly. There was no splenic enlargement, friction rub, or rebound abdominal tenderness. Leukocytes numbered 24,000 per cu mm with 64 percent eosinophils. Progressive weight loss and daily spiking fevers to 39.8°C (103.5°F) persisted for four weeks despite extensive diagnostic tests that included ten negative examinations for ova and parasites in the stool and a trial of metronidazole.

The patient was transferred to the University of California Hospital in San Francisco. Hemoglobin was 8.7 grams per 100 ml, the hematocrit 26 percent, and leukocyte count 15,900 per cu mm with 68 percent eosinophils. The erythrocyte sedimentation rate was 108 mm in one hour. Total iron-binding capacity was 146 mg per 100 ml with 50 percent saturation. The vitamin B_{12} level was 520 picograms per 100 ml. Coombs tests were negative. Bone marrow showed mature eosinophilic hyperplasia with normal iron stores and no granulomas or evidence of metastatic malignant disease or leukemia. Liver function tests yielded the following abnormal values: alkaline phosphatase, 252 international units (IU) per liter (normal, 25 to 80 per liter); and leucine aminopeptidase, 105 IU per liter (normal, up to 45 per liter). Bilirubin, glutamic oxaloacetic transaminase, and lactate dehydrogenase were normal. Skin tests for fungal diseases and tuberculosis were negative, as were serological tests for infection due to Entamoeba and Echinococcus. An upper gastrointestinal series and barium enema studies were negative. A cholecystogram and a cholangiogram showed no calculi and no dilatation of the biliary system.

Multiple focal defects were seen on liver scan

*Experimental drug provided by the Parasitic Disease Drug Service, Parasitic Diseases Branch, Epidemiology Program, Center for Disease Control, U.S. Public Health Service, Atlanta, Ga. 30333.